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| APPLICATION NO. | FILING DATE | FIRST NAMED INVENTOR | ATTORNEY DOCKET NO. | CONFIRMATION NO. |
|---|---|---|---|---|
| 10/048,186 | 06/19/2002 | James C Liao | 06497-013002 | 2905 |

7590 02/25/2004

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| EXAMINER | |
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| ART UNIT | PAPER NUMBER |
|---|---|
| 1652 | |

DATE MAILED: 02/25/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

| Office Action Summary | Application No. | Applicant(s) | |
|---|---|---|---|
| | 10/048,186 | LIAO, JAMES C | |
| | Examiner | Art Unit | |
| | Rebecca E. Prouty | 1652 | |

*-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --*

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

1)☒ Responsive to communication(s) filed on *05 December 2003*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

4)☒ Claim(s) *1-5,7,9-14 and 16-48* is/are pending in the application.

4a) Of the above claim(s) *11,14,22,23,28,32,37-39 and 46-48* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-5,7,9,10,12,13,16-21,24-27,29-31,33-36 and 40-45* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

**Application Papers**

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *10/02, 5/03, 7/03*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

Claims 6, 8, and 15 have been canceled. Claims 1-5, 7, 9-14, 16-35, and new claims 36-48 are at issue and are present for examination.

Applicant's election with traverse of Group I, Claims 1-21, 24-35 and new claims 36-45 and of *glnAp2* as promoter species and isopentenyl diphosphate isomerase (idi) as heterologous polypeptide in the response filed 12/5/03 is acknowledged. The traversal is on the ground(s) that PCT Rule 13.1 does not require nor authorize election of a species in the singular and narrow sense of a requirement for species election under 37 C.F.R. 1.141 because a species is not a general inventive concept. This is not found persuasive because the phrase "single general inventive concept" recited in PCT Rule 13.1 is a requirement for unity of invention. As stated in 37 CFR 1.475 "the requirement of unity of invention shall be fulfilled only when there is a technical relationship among those inventions involving one or more of the same or corresponding special technical features. The expression "special technical features" shall mean those technical features that define a contribution which each of the claimed inventions, considered as a whole, makes over the prior art". As further explained in MPEP 1850 for claims in genus/species relationship "If the independent claims avoid the prior art and satisfy the

requirement of unity of invention, no problem of lack of unity arises in respect of any claims that depend on the independent claims. In particular, it does not matter if a dependent claim itself contains a further invention. Equally, no problem arises in the case of a genus/species situation where the genus claim avoids the prior art. Moreover, no problem arises in the case of a combination/subcombination situation where the subcombination claim avoids the prior art and the combination claim includes all the features of the subcombination. If, however, an independent claim does not avoid the prior art, then the question whether there is still an inventive link between all the claims dependent on that claim needs to be carefully considered. If there is no link remaining, an objection of lack of unity (that is, arising only after assessment of the prior art) may be raised. Similar considerations apply in the case of a genus/species or combination/subcombination situation. In the instant case the generic claims linking the claimed species lacks unity of invention as it does not define a contribution over the art as Haldimann et al. teach an E. coli comprising a *VanH* promoter fused to a *lacZ* gene as well as a *VanS* mutation which inactivates the *VanS* histidine kinase. This fusion is regulated by the

presence of *VanR* and acetyl phosphate levels. Furthermore, the claimed species do not have any shared technical feature at all.

The requirement is still deemed proper and is therefore made FINAL.

Claims 11, 14, 22, 23, 28, 32, 37-39 and 46-48 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention (Claims 22, 23 and 46-48) or species (Claims 11, 14, 28, 32, and 37-39), there being no allowable generic or linking claim. Note Claims 11 and 28 correspond to a non-elected species of gene as the metabolite synthesized by the elected idi gene is dimethylallyl diphosphate (DMAPP) which is not a carotenoid as required by these claims.

Claim 25 is objected to because of the following informalities: the word "that" in line 2 should be replaced with "the". Appropriate correction is required.

Claims 1-5, 7, 9, 10, 12, 13, 16-21, and 36 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 7 is incomplete as dependent on canceled Claim 6. For purposes of examination, this claim is interpreted as if dependent from Claim 1.

Claims 1 and 9 (upon which Claims 2-5, 7, 10-13, 16-21 and 36 depend) are indefinite in the recitation of "heterologous metabolite". The specification on page 1 states "the term "heterologous" refers to a polypeptide or metabolite which is introduced by artifice. **A heterologous polypeptide or metabolite can be identical to endogenous entity that is normally present.**". This definition is contrary to the accepted meaning in the art of the term. The skilled artisan would define the term "heterologous metabolite" as a metabolite not produced by the host cell and thus would take this term as excluding compounds endogenously produced by the host. While applicants may be their own lexicographer, use of terminology repugnant to the ordinary meaning of a term in the art renders the claims confusing and indefinite. It is suggested that the word "heterologous" be deleted.

Claim 9 (from which claims 10, 12, 13, and 16-21 depend) is confusing in the recitation of "enzyme that catalyzes biosynthesis of a heterologous metabolite ... and ... other enzymes that catalyze biosynthesis of the metabolite" as this appears to recite a cell encoding two different enzymes that catalyze the synthesis of the same metabolite by different reactions (for example phosphoenol pyruvate synthase and enolase

both of which produce PEP) but applicants intent instead appears to be to recite a cell encoding two enzymes which catalyze separate reactions in a biosynthetic pathway for a single desired compound.

Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29, 30, 33-36, and 40-45 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

These claims are directed to a genus of bacterial host cells, kits comprising said host cells or nucleic acid constructs for producing such host cells wherein the host cells comprise any promoter which is controlled by any response regulator protein/heterologous polypeptide construct and any genetic modification that makes the promoter regulated by acetyl phosphate or a metabolite whose concentration is indicative of availability of any precursor for a metabolite produced by the heterologous polypeptide. The specification teaches only five representative species of such host cells and constructs, i.e., *E. coli* strains JCL1596, BW18302(*glnAp2-aroG*), BW18302(*glnAp2-pps*), BW18302(p2IDI), and BW18302(p2IDI/pPSG184) all of which

utilize the same bacterial host cell with the same genetic modification (*E. coli* having a *glnL* deletion mutation) and the same promoter which is regulated by the same response regulator which is responsive to the same metabolite (*glnAp2* which is regulated by *ntrC* and responsive to acetyl phosphate levels). Moreover, the specification fails to describe any other representative species by any identifying characteristics or properties. Given this lack of description of representative species encompassed by the genus of the claim, the specification fails to sufficiently describe the claimed invention in such full, clear, concise, and exact terms that a skilled artisan would recognize that applicants were in possession of the claimed invention.

Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29, 30, 33-36, and 40-45 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for *E. coli* having an inactivating *glnL* mutation which are transformed with a nucleic acid encoding a heterologous polypeptide operably linked the *glnAp2* promoter, kits comprising an *E. coli* having a *glnL* mutation and a nucleic acid encoding the *glnAp2* promoter or nucleic acid constructs therefore, does not reasonably provide enablement for any host cell wherein the host cell comprises any

promoter which is controlled by any response regulator protein/heterologous polypeptide construct and any genetic modification that makes the promoter regulated by acetyl phosphate or a metabolite whose concentration is indicative of availability of any precursor for a metabolite produced by the heterologous polypeptide or nucleic acid constructs therefore. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

These claims are so broad as to encompass any bacterial host cell or kits comprising said host cells wherein the host cells comprise any promoter which is controlled by any response regulator protein/heterologous polypeptide construct and any genetic modification that makes the promoter regulated by acetyl phosphate or a metabolite whose concentration is indicative of availability of any precursor for a metabolite produced by the heterologous polypeptide and nucleic acid constructs for producing such host cells. The scope of each of these claims is not commensurate with the enablement provided by the disclosure with regard to the extremely large number of promoters, regulatory proteins, metabolites whose concentration is indicative of availability of any precursor for a metabolite

produced by any heterologous polypeptide and genetic modifications of the host broadly encompassed by the claims. The amino acid sequence of a protein determines its structural and functional properties and likewise the nucleotide sequence of a promoter determines its properties. Thus, predictability of which promoters, regulatory proteins and genetic modifications within a bacterial host can be tolerated and still have the desired ability to regulate function in response to a particular compound, requires a knowledge of and guidance with regard to (i) which promoters are regulated by what proteins, (ii) which amino acids/nucleotides in the sequence of the promoters and regulatory proteins are tolerant of modification and which are conserved (i.e. expectedly intolerant to modification), (iii) a detailed knowledge of the ways in which each promoter/regulatory proteins' structure relates to its ability to regulate transcription (iv) what metabolites levels are reflective of the levels of other metabolites under what conditions, and (v) how the structure and function of each of these is influenced and modified by potential modifications of the cell in which it occurs. However, in this case the disclosure is limited to the use of *E. coli* strains JCL1596, BW18302(*glnAp2-aroG*), BW18302(*glnAp2-pps*), BW18302(p2IDI), and BW18302(p2IDI/pPSG184) all of which utilize

the same bacterial host cell with the same genetic modification (*E. coli* having a *glnL* mutation) and the same promoter which is regulated by the same response regulator and response to levels of the same metabolite (*glnAp2* which is regulated by *ntrC* and responsive to acetyl phosphate levels).

The specification does not support the broad scope of Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29, 30, 33-36, and 40-45, because the specification does not establish: (A) the identity of promoters, regulatory proteins and genetic modifications within a bacterial host that have the desired ability to regulate function in response to acetyl phosphate or other metabolites; (B) regions of any promoter/regulatory protein pair which may be modified without effecting the activity of said promoter to activate transcription in response to the regulatory protein and how the activity of any promoter/regulatory protein pair is influenced by the genetic modification of the host cell itself; (C) the general tolerance of the activities of said promoter/regulatory proteins to modification and extent of such tolerance; (D) a rational and predictable scheme for choosing which promoter/regulatory proteins to screen for the recited utilities and what types of bacterial cell modifications will produced the desired effects of the alteration of these

functions; (E) a rational and predictable scheme for modifying any gene of any bacteria with an expectation of obtaining the desired biological function; and (F) the specification provides insufficient guidance as to which of the essentially infinite possible choices of promoters, regulatory proteins and metabolites and genetic modifications within a bacterial host is likely to be successful.

Thus, applicants have not provided sufficient guidance to enable one of ordinary skill in the art to make and use the claimed invention in a manner reasonably correlated with the scope of the claims broadly including any host cell comprising any promoter which is controlled by any response regulator protein/heterologous polypeptide construct and any genetic modification that makes the promoter regulated by acetyl phosphate or other metabolites. The scope of the claims must bear a reasonable correlation with the scope of enablement (In re Fisher, 166 USPQ 19 24 (CCPA 1970)). Without sufficient guidance, determination of the identity of bacterial cells having the desired biological characteristics is unpredictable and the experimentation left to those skilled in the art is unnecessarily, and improperly, extensive and undue. See In re Wands 858 F.2d 731, 8 USPQ2nd 1400 (Fed. Cir, 1988).

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless -
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1 and 2 are rejected under 35 U.S.C. 102(b) as being anticipated by Haldiman et al. (Reference AK of applicant's PTO-1449).

Haldiman et al. teach an *E. coli* strain BW24386 which comprises a nucleic acid encoding a *vanH* promoter operably linked to a *lacZ* gene (which encodes β-galactosidase which produces galactose as metabolite) and comprising *ackA*, Δ*phoR*, and Δ*creC* mutations such that the *vanH* promoter is regulated by acetyl phosphate. This strain anticipates Claims 1 and 2.

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1-5, 7, 9, 16-20, 24-26, 30, 31, 34, 36, and 42-45 are rejected under 35 U.S.C. 103(a) as being unpatentable over Liao (WO96/08567) in view of Bock et al. (US Patent 5,830,692),

McCleary et al. (Reference AL of applicant's PTO-1449), McCleary et al. (Reference AM of applicant's PTO-1449) and Haldiman et al. (Reference AK of applicant's PTO-1449) or Feng et al. (Reference AJ of applicant's PTO-1449).

Liao teach constructs for the recombinant expression of phosphoenol pyruvate synthase (pps) in cells producing aromatic metabolites and that the increased expression of pps is useful for increasing the amount of carbon flow into the aromatic pathway by producing increased amounts of DAHP. The constructs of Liao comprise the pps gene under the control of an inducible promoter. (see pages 18-19). Liao further shows that cells lacking induction of the pps gene produce significant amounts of the fermentation byproduct acetate indicating significant flux away from PEP and the aromatic pathway (see pages 22-23) but that induction of the pps gene produces undetectable levels of acetate in the cells and near theoretical yields of DAHP.

Bock teach that inducible promoters such as lac, tac, and trp promoters possess several disadvantages in relation to their use for industrial production. These are that the repressors and inducers necessary for use of these promoters are expensive and difficult to handle, particularly when they are metabolizable substances (such as lactose and tryptophan), and cannot be

induced completely when the repressor is present in molar excess. (see columns 1-2).

McCleary (AK) and McCleary (AP) teach that acetyl phosphate may act a global regulatory signal in *E. coli* responsible for the activation of a wide range or response regulators of two-component systems, including the *glnAp2* promoter, in the absence of their cognate histidine kinase (i.e., the *ntrB* gene product in the case of *glnAp2*). They further teach that acetyl-phosphate levels in bacteria correlate with the amount of acetate produced.

Haldiman et al. and Feng et al. each teach *E. coli* two-component system promoters (the *VanH* promoter in Haldiman et al. and the *glnAp2* promoter in Feng et al.) which are activated by a response regulator protein (*VanR* in Haldiman et al. and *NtrC* in Feng et al.) and acetyl phosphate in the absence of the corresponding histidine kinases (*VanS* in Haldiman et al. and *NtrB* in Feng et al.) and in the presence or absence of nitrogen.

As inducers (IPTG) for promoters such as *tac* used by Liao are expensive and have disadvantages as taught by Bock, it would have been obvious to one of ordinary skill in the art to link the production of pps to the presence of a metabolite in the cell which signals that significant amounts of carbon are being diverted away from the aromatic biosynthetic pathway. Liao teach

that acetate production occurs under these conditions. Therefore, it would have been obvious to one of ordinary skill in the art to replace the *tac* promoters in the constructs of Liao with a promoter which is induced by high acetate levels. As McCleary et al. (AK and AP) teach that acetyl-phosphate levels correlate with the amount of acetate produced, it would have been obvious to one of ordinary skill in the art to link the pps gene to the acetyl-phosphate regulated promoters taught by Haldiman et al. or Feng et al. and express these constructs in *E. coli* cells which lack the cognate histidine kinases such that the response regulators which activate transcription from these promoters are activated by acetyl phosphate. Furthermore, it would have been obvious to one of ordinary skill in the art to put the cells and vectors necessary for production or high levels of aromatic metabolites together in a kit for easy handling.

Claims 1-5, 7, 9, 10, 12, 13, 16-20, 24-27, 29-31, 33-36, and 40-45 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kajiwara et al. (1997) in view of Bock et al. (US Patent 5,830,692), McCleary et al. (Reference AL of applicant's PTO-1449), McCleary et al. (Reference AM of applicant's PTO-1449) and Haldiman et al. (Reference AK of

applicant's PTO-1449) or Feng et al. (Reference AJ of applicant's PTO-1449).

Kajiwara et al. teach constructs for the recombinant expression of isopentenyl diphosphate isomerase (idi) in *E. coli* cells producing carotenoid metabolites and that the increased expression of idi is useful for increasing the amount of carotenoids produced. The constructs of Kajiwara et al. comprise the idi gene under the control of the inducible lac promoter. (see Fig 1). Kajiwara et al. further show the metabolic pathway for the production of carotenoids in *E. coli* and show that the initial precursors of all carotenoids are the glycolytic intermediates acetyl-CoA, pyruvate and glyceraldehyde 3-phosphate (GAP) (see page 422).

Bock teach that inducible promoters such as lac, tac, and trp promoters possess several disadvantages in relation to their use for industrial production. These are that the repressors and inducers necessary for use of these promoters are expensive and difficult to handle, particularly when they are metabolizable substances (such as lactose and tryptophan), and cannot be induced completely when the repressor is present in molar excess. (see columns 1-2).

McCleary (AK) and McCleary (AP) teach that acetyl phosphate may act a global regulatory signal in *E. coli* responsible for the activation of a wide range or response regulators of two-component systems, including the *glnAp2* promoter, in the absence of their cognate histidine kinase (i.e., the *ntrB* gene product in the case of *glnAp2*). They further teach that acetyl-phosphate levels in bacteria correlate with the amount of acetyl-CoA produced and is present at high levels whenever glycolytic intermediates accumulate.

Haldiman et al. and Feng et al. each teach *E. coli* two-component system promoters (the *VanH* promoter in Haldiman et al. and the *glnAp2* promoter in Feng et al.) which are activated by a response regulator protein (*VanR* in Haldiman et al. and *NtrC* in Feng et al.) and acetyl phosphate in the absence of the corresponding histidine kinases (*VanS* in Haldiman et al. and *NtrB* in Feng et al.) and in the presence or absence of nitrogen.

As inducers (IPTG) for promoters such as *lac* used by Kajiwara et al. are expensive and have disadvantages as taught by Bock, it would have been obvious to one of ordinary skill in the art to link the production of idi to the presence of a metabolite in the cell which signals that significant amounts of the precursors for carotenoid biosynthesis are present. McCleary et

al. (1993) teach that acetyl phosphate accumulation occurs under these conditions (i.e., accumulation of the glycolytic intermediates that are the immediate precursors of carotenoid biosynthesis). Therefore, it would have been obvious to one of ordinary skill in the art to replace the *lac* promoters in the constructs of Kajiwara et al. with a promoter which is induced by high acetyl phosphate levels. As McCleary et al. (AK and AP) teach that acetyl-phosphate levels correlate with the amount of glycolytic intermediates produced, it would have been obvious to one of ordinary skill in the art to link the idi gene to the acetyl-phosphate regulated promoters taught by Haldiman et al. or Feng et al. and express these constructs in *E. coli* cells which lack the cognate histidine kinases such that the response regulators which activate transcription from these promoters are activated by acetyl phosphate. Furthermore, it would have been obvious to one of ordinary skill in the art to put the cells and vectors necessary for production of high levels of carotenoids together in a kit for easy handling.

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982);

*In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970);and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29-31, 33-35, and 40-45 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 36-40 of copending Application No. 09/626,612. An obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but an examined application claim not is patentably distinct from the reference claim(s) because the examined claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985). Although the conflicting claims are not identical, they are not patentably distinct from each other because Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29-31, 33-35, and 40-45 are generic to all that is recited in claims 36-40 of copending

application 09/626,612. That is, claims 36-40 of copending application 09/626,612 fall entirely within the scope of Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29-31, 33-35, and 40-45 or, in other words, Claims 1-5, 7, 9, 10, 12, 13, 16-21, 24-27, 29-31, 33-35, and 40-45 are anticipated by claims 36-40 of copending application 09/626,612.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Rebecca Prouty, Ph.D. whose telephone number is (571) 272-0937. The examiner can normally be reached on Monday-Friday from 8:30 to 4:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ponnathapura Achutamurthy, can be reached at (571) 272-0928. The fax phone number for this Group is (703) 872-9306.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (571) 272-1600.

Rebecca Prouty
Primary Examiner
Art Unit 1652